

There are perhaps other reasons why a disease that sometimes occurs in bunches in a span of time at one place may at other places appear with some regularity. After reviewing the little information that exists on the epidemiology of TEF and/or OA (TEF/OA) and after comparing the patterns of TEF/OA with other congenital abnormalities, we feel that clusters in time and space do occur and in this paper shall present some statistical rationale to support this. The table below consists of incidence data of births with TEF/OA as reported in investigations previously cited.

Predicted and Observed Cluster Sizes of Congenital
Tracheoesophageal Fistula and Oesophageal Atresia

<u>Source</u>	<u>Consecutive Annual Incidence</u>	<u>Actual Cluster Size</u>	<u>Maximum as Predicted from Cluster Model</u>	<u>Maximum Predicted if No Clustering</u>
<u>BIRMINGHAM</u>				
1950-55 Knox	2, 2, 2, 15, 5, 9	15	15	<10
<u>NEWCASTLE</u>				
1950-58 Knox	1, 8, 1, 4, 3 16, 11, 7, 12	8 16	9 24	6 13
<u>PENNSYLVANIA*</u>				
1951-58 Babbott, Ingalls	5, 13, 6, 0 7, 7, 2, 20	13 20	13 19	7 10

*These data represent only TEF.

Grimson (6) has developed a cluster model that appears appropriate, both in terms of rationale and fit, in describing infectious processes. By considering the span of years to be 4, 5 or 6 (this is a parameter in the model called the cluster modulus, which is similar to the idea of periodicity), the actual maximum cluster size is, with good accuracy, predicted by the cluster model. In general, it significantly exceeds the estimates from the Ederer-Myers-Mantel model which produces maximum numbers under the assumption of no clustering; this provides a statistical test for clustering (7,8).

Viral etiology has been implicated in some anomalies; in particular, it is widely accepted that rubella, maternal cytomegalovirus and coxsackieviruses act on the fetus. Other infectious diseases including hepatitis, mumps and influenza have been suggested as correlates of congenital anomalies but little or no hard evidence exists, especially in connection with TEF/OA.

Few epidemiological attempts have been made to try to link influenza with TEF, OA and other anomalies. In addition to providing a quantitative logic and description behind the cluster question, we shall offer a conjecture that women who are in their first trimester of pregnancy during a period of a high incidence of influenza A are at higher risk of giving birth to a child with TEF/OA than are women in early stages of pregnancy at other times.